LIVER RUPTURE IN PREGNANCY : A TYPICAL CASE?

H Cheung, H Hamzah

ABSTRACT:

We report a patient who presented with abruptio placenta and hepatic rupture at 30 weeks gestation. The latter is a recognised but rare complication of pre-eclampsia and eclampsia. The typical features of this entity as described in the literature and as seen in this patient are emphasized as increased awareness can lead to early diagnosis and better prognosis.

Keywords: Hepatic haemorrhage, rupture, pregnancy, eclampsia

SINGAPORE MED J 1992; Vol 33: 89-91

INTRODUCTION

Hepatic haemorrhage and rupture during pregnancy is a rare but serious complication of pre-eclampsia and eclampsia. This entity was first reported in 1844 by Abercrombie [1]. Fetal and maternal mortality is about 60%, even with surgical treatment [2]. Maternal mortality of 96% is reported with conservative treatment[3].

The typical patient is multiparous, in her late 20's or 30's and presents in the 3rd trimester of pregnancy. The entity is said to be biphasic with a prodrome of epigastric and right upper quadrant (RUQ) discomfort accompanying pre-eclampsia. The second phase of actual liver rupture is heralded by sudden severe pain and rapidly ensuing signs of hemoperitoneum and shock [3].

We report a patient who presented acutely with impending shock, abruptio placenta, intrauterine fetal death and liver haemorrhage and rupture. The purpose of this report is to increase awareness of this entity amongst clinicians, so that early diagnosis may be made.

CASE HISTORY

A 29-year-old Malay lady, G3P2, presented at 30 weeks gestation, as an emergency to the Subang Jaya Medical Centre on 30 August 1985 with an acute history of sudden severe abdominal pain. She had a history of eclampsia with seizures complicating 2 previous pregnancies. The first resulted in a livebirth, while the second resulted in a fresh stillborn at Caesarean section. Her present antenatal care had been at another hospital where she had been booked for delivery.

On physical examination, she was pale and in pain with a tachycardia of 120/min and a Bp of 90/60. The abdomen showed the presence of a lower paramedian scar and an enlarged uterus, and was tense to palpation with rebound tenderness. Ultrasound by the attending obstetrician revealed abruptio placenta. As this was thought to be sufficient cause for her clinical state, the patient was rushed to surgery. Urgent blood tests showed the following: Hb 9.2 gm/dL (Normal 11.5 - 16), PCV 29% (Normal 37 - 47%), Platelets 57 x 10^9/L (Normal 150 - 400), Blood sugar 6.6 mmol/L (Normal 4.4 -6.7), creatinine 146 umol/L (Normal 44 - 107), SGOT 400 IU/L (Normal 0 - 58), SGPT 489 IU/L (Normal 0 - 58), SAP 121 IU/L (Normal 40 - 136), Bleeding time 9 min 39.5 sec (Normal 2 - 5 min), Clotting time 11 min 49 sec (Normal 4 - 10 min), Prothrombin time 22.8 sec (Control 13 sec). APTT was not available.LSCS was performed and a stillborn fetus was delivered. Abruptio placenta was confirmed and blood loss was estimated at 1 1/2 litres.

The following day, signs of continuing bleeding were present despite blood replacement, and a laparotomy was performed. At operation the peritoneal cavity was filled with blood. A ruptured liver with a subcapsular haematoma was found. The uterus showed no rupture or bleeding. The haematoma was evacuated and a branch of the Rt. Hepatic Artery was ligated. Subtotal hysterectomy and left salpingo-oophorectomy were also performed. Her post-operative course was initially stormy; her mean arterial pressure was 120 - 130 mm Hg despite adequate anti-hypertensive treatment. Left basal pneumonia developed which promptly responded to antibiotic therapy with Cefoperazone (Cefobid). Renal and hepatic dysfunction worsened with serum creatinine climbing to 327 umol/L, SGOT to 3500 IU/L and SGPT to 2040 IU/L. She required intensive care and was managed by a multi-disciplinary team of doctors comprising obstetrician, cardiologist and two surgeons. Her treatment included 45 units of whole blood, 10 units of fresh frozen plasma, 2 units of packed cells, antihypertensive agents, diuretics and antibiotics. Clinical improvement was slow but after 4 weeks, her Hb, WCC, SGPT, SGOT, Creatinine and electrolytes returned to normal and she was transferred out of intensive care.

However, a week later, she began to complain of recurrent RUQ pain and developed a low-grade fever. A CXR showed a right pleural effusion. This was confirmed on ultrasound which also revealed a small right subphrenic collection. Aspiration of the pleural effusion yielded 500 cc of straw-coloured fluid. There was reaccumulation of the effusion on a repeat ultrasound 2 days later; the subphrenic collection was larger with septae present. Infected haematoma was suspected, and diagnostic tap revealed altered blood. The haematoma was surgically evacuated. Following this third operation, she improved and became well enough to be discharged home.

Recurrent RUQ pain prompted two further admissions. On the second, CXR showed reaccumulation of the right pleural effusion (Fig 1), and ultrasound further revealed recurrence of a right subphrenic collection, aspiration of which yielded a mixture of blood and pus. CT confirmed the presence of a hypodense subcapsular haematoma on the anterior supero-lateral aspect of the right lobe of liver with bands of varying attenuation thought to be consistent with repeated bleeding or oozing (Fig 2,3). Surgery was again deemed necessary, and at surgery active bleeding was detected in the right lobe of the

Department of Diagnostic Radiology & Organ Imaging
Chinese University of Hong Kong
Prince of Wales Hospital
Shatin, NT
Hong Kong

H Cheung, MBChB(Edin), MSc, DMRD, FRCR
Consultant Radiologist

Dept of Obstetrics & Gynaecology
Subang Jaya Medical Centre
Selangor, Malaysia

H Hamzah, MBBS, MRCOG
Consultant Obstetrician & Gynaecologist

Correspondence to : Dr H Cheung

Fig 1-Chest radiograph showing recurrent right pleural effusion.

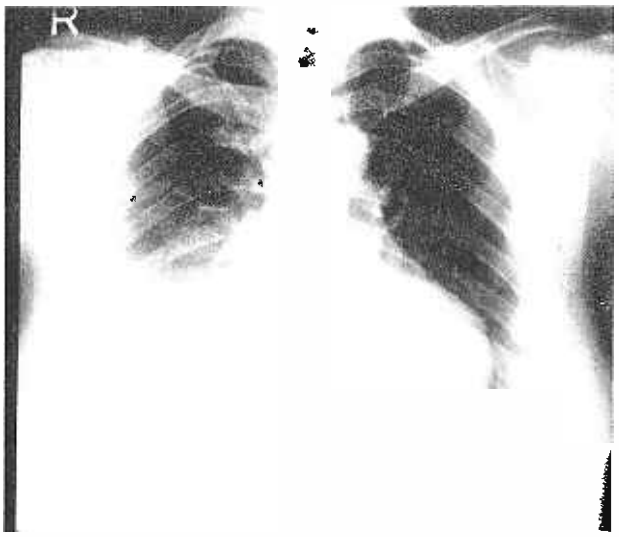


Fig 2-CT image of the upper abdomen showing a subcapsular haematoma along the lateral liver edge (arrow). An oval-shaped liquefying haematoma is present in the adjacent right lobe.

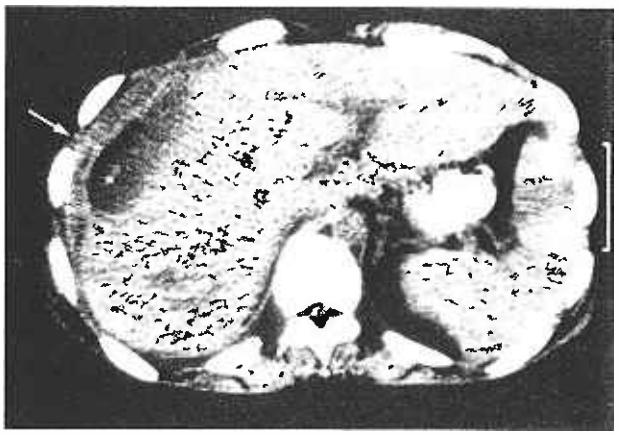
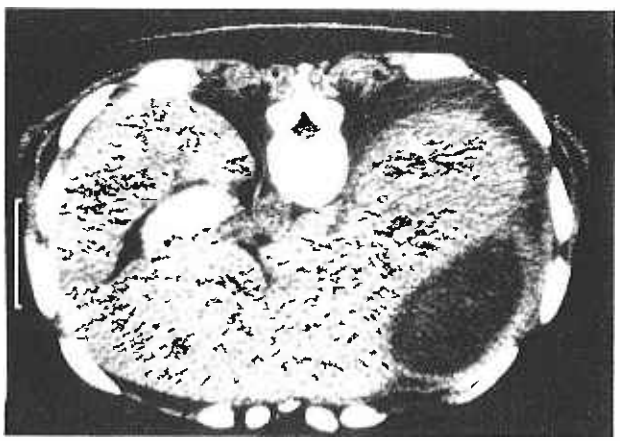


Fig 3- CT image at a higher level showing the largest dimension of the intrahepatic haematoma, surrounded by the subcapsular haematoma. Postero-laterally (arrow), subphrenic and pleural collections obliterate the diaphragmatic crus.



liver. Infected clots were evacuated and a right lobectomy performed.

She developed a fecal fistula 4 weeks post-lobectomy, and an intra-abdominal abscess about 5 months later. Both were successfully treated surgically during short periods of hospital stay. Bleeding from the liver did not recur and she is now completely well.

DISCUSSION

The brain, kidney and liver are the three main target organs damaged in toxaemia of pregnancy. While CNS catastrophies are the main cause of death, hepatic complications such as infarction, haematomas and rupture, contribute to mortality in 16%[4]. Although hepatocellular dysfunction with elevated transaminases is seen in more than half of patients with severe pre-eclampsia and eclampsia[4], life-threatening liver haematomas with rupture is rare[2,3,5], and much less well recognized.

In a review of 75 reported cases of ruptured liver in pregnancy, dating back to the early 1900's, Henny[3] found that the typical patient is multiparous, in her late 20's or 30's and presents most frequently in the third trimester of pregnancy. Rupture in the early postpartum period is also documented[6-10]. The condition shows a characteristic biphasic chronological sequence with a prodrome of epigastric discomfort accompanying hypertension and pre-eclampsia. This can exist as long as a month before the acute symptomatology of liver rupture and shock occurs. As the syndrome is rare and not commonly considered, cases are frequently misdiagnosed pre-operatively as, among others, abruptio placenta, ruptured uterus, perforated ulcer or appendix, myocardial infarction and pulmonary embolus[3].

The majority of haematoma is limited to the anterosuperior aspect of the liver. In 75% only the right lobe is involved, and rupture of Glisson's capsule usually occur at the inferior margin [3]. Although rupture is often spontaneous, it has been suggested that minor exogenous or endogenous trauma unrecognized by the patient may predispose to it[11].

It has been hypothesized that subcapsular haematoma without rupture could be subclinical and have a higher incidence than is presently believed[3]. Manas[9] describes seven patients with haematoma but without rupture which represents about 1% of the total number of patients with pre-eclampsia seen over the same period in the same medical centre. However all of them were symptoma with acute epigastric or RUQ pain, and asymptomatic cases have not been described.

There are many hypotheses as to the pathogenesis of the haematoma and rupture of the liver capsule. Rademaker[11] describes a chain of events which begins with infarction and evolves through hypervascularisation, rupture of vessels, intrahepatic haemorrhage, subcapsular haematoma, rupture of the capsule, haemoperitoneum, and peritonitis which can lead to death. In a study of 102 cases submitted for pathology, Rolfes[4] found that the histopathology of toxaemic liver disease was characterized by variable combinations of periportal fibrin depositions, haemorrhages and hepatocelluar necrosis. He concluded that a toxaemic vasculopathy related to severe vasospasm in the hepatic arterial circulation may be responsible for the changes. Disseminated intravascular coagulation and isolated thrombocytopenia are also thought to contribute towards the tendency to liver haemorrhage and liver damage [3,4].

Laboratory findings of thrombocytopenia, low haemoglobin and hematocrit, abnormal liver and renal function indices are commonly present in these patients[3,4,6,9], including evidence of DIC with elevation of fibrin degradation products[7,9]. The presence of an associated right pleural effusion is also commonly seen[7, 10, 12].

The important contribution of CT[5, 7-10] and ultrasound[6, 8-10] towards early diagnosis and management is well recognised.

Isotope liver scans with Tc-sulphur colloid[7-9] have also been used although results are less specific. Angiography contributes to the diagnosis and identification of the bleeding site[5,7,8]. Transcatheter embolisation has been successful in treatment[7], and embolotherapy is likely to feature more in the future.

We wish to highlight the typical features in our patient. She is in her late twenties, multiparous and presented acutely with severe abdominal pain and impending shock, in the second phase of the condition according to Henny[3]. A clear history of eclampsia during a previous pregnancy was present. Blood tests revealed low platelet counts and deranged liver function tests. Her initial presentation was attributed to abruptio placenta, and this association has been described[2]. A missed diagnosis due to failure to examine the liver sonographically has been observed before[5]. The site of haemotoma in this patient is the commonest reported in the literature[3].

A prolonged and complicated hospitalization is reported after surgery for liver repair and drainage of the haematoma[2,6], with average hospital stay of 31 days. This lends support to the argument for conservative treatment which is reported to be successful[5,6,9,10]. This consists of blood replacement, correction of coagulation defects, early termination of pregnancy and monitoring of respiratory function and status of the haematoma by ultrasound monitoring[5-10]. Following rupture, it is recommended that ligation of bleeding points and of the Hepatic Artery should be performed prior to lobectomy[10]. These were observed in the management of our patient. Despite this, bleeding recurred and right lobectomy was performed. She experienced a prolonged period of hospitalization and ill health which lasted seven months. However she is now completely recovered, and is well at the time of writing.

CONCLUSION

Toxaemic liver haematoma and rupture in eclampsia of pregnancy is rare. Treatment is controversial, with mandatory surgery advocated by some[3,13], and conservative treatment by others[5-10].

Individual prognosis is dependent on time of diagnosis and subsequent therapy[3]. Early diagnosis may only be made with increased awareness of this entity. In this case report we have highlighted the typical features, and recommend that the liver should be examined carefully during ultrasound of the eclamptic patient with abdominal pain.

ACKNOWLEDGEMENTS

The authors wish to thank Miss Sanny Chan for typing the manuscript and the Medical Media Services, PWH, for preparing the photographs.

REFERENCES

1. Abercrombie J : Haemorrhage of the liver. London Medical Gazette 1844; 34: 792-4.
2. Bis KA, Waxman B: Rupture of the liver associated with pregnancy: A review of the literature and report of 2 cases. Obstet Gynecol Surg 1976; 31: 763-73.
3. Henny CP, Lim AE, Brummelkamp WH, Buller HR, Ten Cate JW. A review of the importance of acute multidisciplinary treatment following spontaneous rupture of the liver capsule during pregnancy. Surg Gynecol Obstet 1983; 156: 593-8.
4. Rolfes DB, Ishak KG: Liver disease in toxaemia of pregnancy. Am J Gastroenterol 1986; 81:1138-44.
5. Ekberg H, Leyon J, Jappsson B et al. Hepatic rupture secondary to Pre-Eclampsia - Report of a case treated conservatively. Annales Chirurgiae et Gynaecologiae 1984; 73: 350-3.
6. Goodlin RC, Anderson JC, Hodgson PE: Conservative treatment of liver hematoma in the postpartum period. A report of two Cases. J Reprod Med 1985; 30: 368-70.
7. Loevinger EH, Vujic I, Lee WM, Anderson MC: Hepatic rupture associated with pregnancy: treatment with transcatheter embolotherapy. Obstet Gynecol 1985; 65: 281-4.
8. Winer-Muram HT, Muram D, Salazar J, Massie JD: Hepatic rupture in Preeclampsia: the role of diagnostic imaging. J Can Assoc Radiol 1985; 36: 34-6.
9. Manas KJ, Welsh JD, Rankin RA, Miller DD: Hepatic Hemorrhage without rupture in Preeclampsia. N Engl J Med 1985; 312: 424-6.
10. Herbert WNP, Brenner WE: Improving survival with liver rupture complicating pregnancy. Am J Obstet Gynecol 1982; 142: 530-4.
11. Rademaker L: Spontaneous rupture of liver complicating pregnancy. Ann Surg 1943; 118: 396-401.
12. Nelson EW, Archibald L, Albo D: Spontaneous hepatic rupture in pregnancy. Am J Surg 1977; 134:817-20.
13. Stalter KD, Sterling WA: Hepatic subcapsular haemorrhage associated with pregnancy. Surgery 1985; 98: 112-3.